

Conventional Endodontic Therapy of Upper Central Incisor Combined with Cyst Decompression: A Case Report

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Abstract

Treatment of a maxillary central incisor with an associated cystic lesion by conventional endodontic therapy combined with decompression is reported. Although small cystic lesions frequently heal simply with endodontic therapy, larger lesions may need additional treatment. If surgical enucleation is elected, other teeth or structures may be damaged unnecessarily. Therefore, a case can be made for first attempting the more conservative treatment of decompression, and a workable protocol for this is presented. In this case, 6 weeks with latex tubing in place and daily irrigation with 0.12% chlorhexidine led to complete healing with no need for further surgery or other root canal therapy on teeth initially surrounded by this lesion. At the 2-year recall, the lesion has completely resolved, and the adjacent teeth remain vital and normal. (*J Endod* 2007; 33:753–757)

Key Words

Decompression, marsupialization, radicular cyst

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Treatment of teeth with large cystic lesions can be problematic. For many years, it was thought that the distribution of cysts among periapical lesions was around 40% to 50% (1, 2). Subsequent studies in which complete lesions were evaluated by serial sectioning revealed that, in fact, many fewer lesions were actually cystic than previously thought (3–6). Of the 15% of lesions now considered to be truly cystic, somewhat more than half are true apical cysts (3–6). According to Nair, "...a periapical pocket cyst may heal after conventional root canal therapy whereas an apical true cyst is less likely to be resolved without surgical intervention..." (7). Treatment of suspected cysts, therefore, requires follow-up over a period of time. According to Natkin et al. (8), there is no doubt that larger lesions are more likely to be cysts and will be less likely to heal with endodontic therapy alone.

Should surgical intervention become necessary, the decision point is whether to raise a flap and completely enucleate the lesion or to try "decompression" first (8–10). Even if enucleation is still necessary later, the lesion will predictably be much smaller and present less difficulty with removal and less risk of damage to associated teeth and vital structures. As an aside, decompression with placement of tubing to maintain drainage is quite different from "marsupialization," although the terms tend to be used interchangeably. Marsupialization is actually described as "...unroofing the outer wall of the cyst by making a surgical incision, evacuating its contents, and establishing a large permanent opening by suturing the remaining part of the cystic membrane to the mucosal surface around the periphery of the opening" (9). Decompression is favored because of lower morbidity and the fact that bony ingrowth occurs as the lesion shrinks in size, thus resulting in more normal bony contours after treatment is concluded.

Case Report

A healthy 13-year-old male was referred for evaluation based on the presence of a sinus tract stoma on the buccal attached gingiva over the left upper lateral incisor. The patient was asymptomatic and not aware of the stoma. A radiograph revealed part of a radiolucent lesion that appeared to be centered on tooth #10. The sinus tract was traced with gutta percha and another radiograph made so that both the full extent of the lesion could be assessed and to see where the sinus tract may have originated (Fig. 1). Sinus tract tracing may or may not reveal anything that would not be revealed with subsequent tests and examination. However, it is one more corroborating bit of evidence, is easy to perform, and carries essentially no risk. The tracing pointed to tooth #9 as the source of the lesion.

Tooth #9 was indeed found to be nonvital (no response to cold or electrical stimulation), whereas all other maxillary anterior teeth were normal in all regards. In consultation with the patient's parents, initial treatment was to be root canal therapy with an interim dressing of calcium hydroxide for a period of 4 weeks. On opening, copious drainage of very thin viscosity was noted, which persisted for some time despite repeated drying with paper points after shaping and alternately irrigating with sodium hypochlorite (NaOCl) 5%, EDTA 17%, and pure ethanol as a final rinse. Vitapex (Dia-Dent Group International, Burnaby, BC, Canada) $\text{Ca}(\text{OH})_2$ paste was injected into the canal space (Fig. 2). Vitapex is $\text{Ca}(\text{OH})_2$ in a silicone base with potassium iodide incorporated as an antimicrobial agent.

At 1 month, the sinus tract was resolved so the $\text{Ca}(\text{OH})_2$ paste was removed with a combination of irrigation as noted earlier and reinstrumentation to slightly larger apical diameter (.070 mm) and taper (12%) using stainless steel hand files and greater taper rotary files. At this visit, complete drying of the canal space was achieved, and the canal

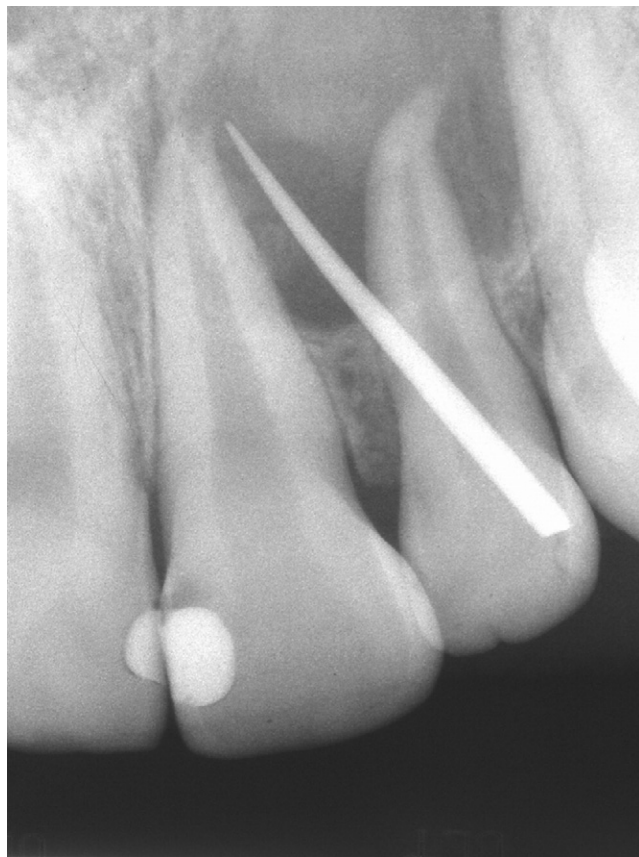


Figure 1. Sinus tract tracing.

was obturated with warm vertical compaction of gutta-percha and AH Plus sealer (Dentsply Maillefer, Ballaigues, Switzerland) (Fig. 3).

At the 3-month recall, the patient was still asymptomatic; however, the sinus tract stoma was present again. At this time, discussion was undertaken with several endodontic specialists, and decompression was considered as a possible treatment alternative. In consultation with the patient and his parents, it was believed that decompression for a period of 6 weeks offered a reasonable chance of success and that even if complete healing did not ensue, enucleation of the residual (smaller) lesion would be less likely to cause damage to adjacent structures, including teeth #10 and 11 and the floor of the nose.

After infiltration anesthesia, an approximately 1.5-cm vertical incision was made in between the root eminences of teeth #9 and 10. Upon entry into the cyst cavity, there was slight drainage of pus followed by copious drainage of the typical straw-colored fluid associated with cystic lesions. Lavage with sterile saline was accomplished, and then an approximately 2-cm length of #10Fr radiopaque latex tubing was inserted to the depth of the cyst cavity. One 4-0-gut suture was placed above and below the drain. Another suture was placed through the drain itself as well as through mucosa to stabilize it during initial healing. At 2 weeks, healing was complete, and the drain could be removed and reinserted with minimal discomfort to patient (Figs. 4, 5). The patient was instructed to irrigate through the lumen of the drain daily with chlorhexidine 0.12%, consistent with a protocol previously described by Brondum and Jensen (11). Their recommendation is once daily with 0.12% chlorhexidine gluconate delivered through "...a normal syringe and a thin blunt hypodermic needle." Irrigation is accomplished through the lumen of the drain. Internal diameter of size #10Fr drain is about 1.5 mm so #16- or 18-gauge blunt Luer lock dispensing tips work

well with small disposable syringes; both are available from any dental supplier. Although empirical, it seems prudent to irrigate with an antibacterial agent, and chlorhexidine has proved safe and effective as a pre-/postsurgical oral disinfectant for many years.

At 6 weeks, the drain was removed with instructions to continue irrigating as the aperture healed. Two weeks later, healing was nearly complete with a very small residual lumen present (Fig. 6). The patient was recalled at 3-month intervals with complete healing noted by 2 years and maintenance of normal pulpal responses in teeth #10 and 11 (Figs. 7, 8).

Discussion

Cyst formation is preceded by periapical granuloma, which represents a host response to inflammation and subsequent infection of the tissues occupying the pulp space. Granulomas comprise "a mixed infiltrate of T and B lymphocytes, PMNs, macrophages, plasma cells, NK cells, eosinophils, and mast cells..." (12). In the rat model, it was shown that pulp infection led to a "...rapid period of lesion expansion and bone destruction...between days 1 and 15 after exposure (active phase), with a chronic phase characterized by lesion stabilization commencing thereafter..." (13). This bone destruction has been shown to be caused by a complex interaction of many mediators, both bacterial-derived (such as lipopolysaccharide) and host-derived (interleukins, tumor necrosis factors, and prostaglandins) (14).

Proliferation of epithelium within a granuloma has long been known to be a response by the cell rests of Malassez to inflammation (15–17). What is less clear is the mechanism by which the cavity of the cyst comes into being. The "nutritional deficiency theory" postulates that the connective tissue surrounding a ball of proliferating epithelium



Figure 2. Vitapex root dressing.



Figure 3. Warm vertical compaction.

supplies its nutrition. If the epithelial proliferation proceeds too fast, the central cells within this ball of epithelium will degenerate and die, leading to a fluid-filled cavity within the epithelial mass (16). The “abscess theory” supposes that proliferation of epithelium traps various connective tissue elements (including inflammatory cells) that then necrose and lead to proteolytic activity, ultimately resulting in the fluid-filled cavity typical of a cyst (17). The fact that no more modern theories (or definitive proof for either of the previously mentioned theories) exist 30 years later is a testament to the complexity of the cellular biology and biochemistry relating to these lesions.



Figure 4. Radiopaque latex drain.

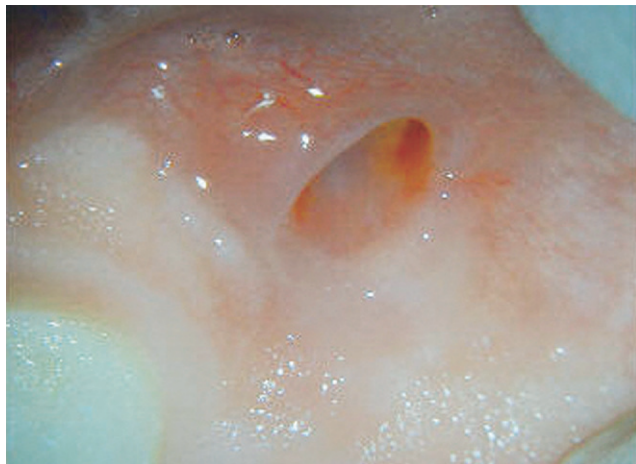


Figure 5. Drain removed.

Once the cystic cavity is formed, then the question is how the third phase (enlargement) progresses. Evidence for a “...molecular explanation...” (5) is building as we add to our understanding of cytokines such as interleukins (ILs), tumor necrosis factors, neuropeptides, and effector molecules such as matrix metalloproteinases. Although intracystic pressure has long been postulated as a reason for cyst expansion, only recently have clues come to light about the possible mechanisms for this. Very recent research suggests that IL-1 alpha expression might be partly regulated by intracystic pressure (18). IL-1 alpha has many functions including induction of osteoclast formation and stimulation of prostaglandin and collagenase production (19). Therefore, it is very likely that reduction of intracystic pressure is a key factor. It is also plausible that reduction of the concentration of inflammatory mediators by irrigation of the cyst lumen could reduce epithelial proliferation and reverse bone resorption, leading to shrinkage of the cyst cavity. However, the exact mechanisms of cyst expansion and shrinkage remain unknown at this time.

Although it is not known what percentage of radicular cyst cases can be expected to heal with only decompression, it is a viable treatment modality that bears consideration when treating large, presumptively cystic lesions. The literature is sparse on this topic. Some case reports show complete healing with no subsequent enucleation (9, 10). Others

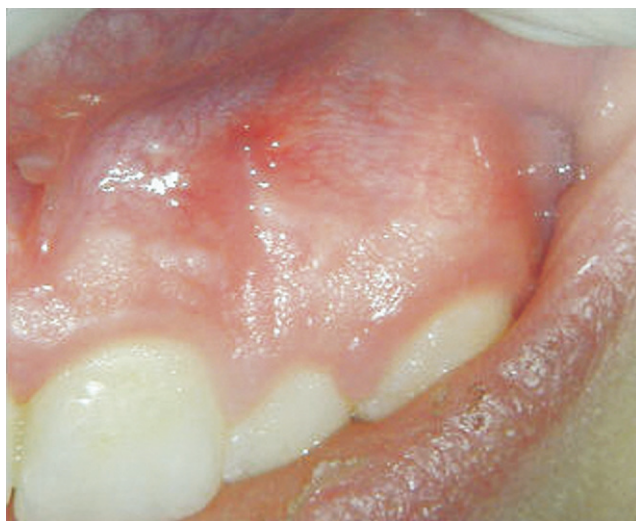


Figure 6. Two weeks after drain removal.

report a secondary surgery for enucleation of the residual (albeit much smaller) cyst. These latter case reports involved extensive lesions in the mandible (20, 21).

In fact, even more aggressive lesions such as odontogenic keratocysts are more often being first treated with decompression and then further treated with enucleation when the lesion is smaller and lining epithelium thicker (11, 22). Several recent studies have confirmed the effectiveness of marsupialization/decompression as a preliminary procedure in the treatment of odontogenic keratocysts, with a surprising number of lesions exhibiting complete healing with no further surgical intervention (22–24).

The risks of decompression are minimal, especially compared with the risk of damaging other vital structures with aggressive surgical enucleation. As to the length of time necessary to leave drains in, there is no standard protocol, and, in fact, it may be different for different kinds, sizes, or locations of lesions. As a practical matter, many patients may not be willing to undergo prolonged treatment of this sort. With complete informed consent, they may prefer more immediate therapy and elect to do enucleation without delay in conjunction with the conventional endodontic therapy. However, most patients prefer the most conservative treatment approach that may lead to healing. This case



Figure 7. Two year recall.



Figure 8. Two year recall.

illustrates the possibility of complete healing of cystic periradicular lesions with a minimally invasive approach.

References

1. Bhaskar SN. Oral surgery-oral pathology conference #17. Walter Reed Army Medical Center. Periapical lesions-types, incidence and clinical features. *Oral Surg* 1966;21: 657–72.
2. Lalonde ER. A new rationale for the management of periapical granulomas and cysts: an evaluation of histopathological and radiographic findings. *J Am Dent Assoc* 1970;80:1056–9.
3. Nair PNR, Pajarola G, Schroeder HE, et al. Types and incidence of human periapical lesions obtained with extracted teeth. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;81:93–102.
4. Simon JHS. Incidence of periapical cysts in relation to the root canal. *J Endod* 1980;6:845–8.
5. Nair PNR. Apical periodontitis: a dynamic encounter between root canal infection and host response. *Periodontology* 2000 1997;13:121–48.
6. Nair PNR. New perspectives on radicular cysts: do they heal? *Int Endod J* 1998;31:155–60.
7. Natkin E, Oswald RJ, Carnes LI. The relationship of lesion size to diagnosis, incidence, and treatment of periapical cysts and granulomas. *Oral Surg* 1984;57:82–94.
8. Freedland JB. Conservative reduction of large periapical lesions. *Oral Surg* 1970;29:455–64.
9. Neaverth EJ, Burg HA. Decompression of large periapical cystic lesions. *J Endod* 1982;8:175–82.
10. Rees JS. Conservative management of a large maxillary cyst. *Int Endod J* 1997;30:64–67.

11. Brondum N, Jensen VJ. Recurrence of keratocysts and decompression treatment. *Oral Surg Oral Med Oral Pathol* 1991;72:265–9.
12. Orstavik D, Pitt Ford TR. *Essential Endodontology*. Malden, PA: Blackwell Publishing, 2002:47.
13. Stashenko P, et al. Kinetics of immune cell and bone resorptive responses to endodontic infections. *J Endod* 1992;18:422–6.
14. Wang CY, Stashenko P. Characterization of bone-resorbing activity in human periapical lesions. *J Endod* 1993;19:107–11.
15. Browne RM. The pathogenesis of odontogenic cysts: a review. *J Oral Pathol* 1975;4:31–46.
16. Shear M. The histogenesis of the dental cyst. *Dent Practitioner* 1963;13:238–43.
17. Summers L. The incidence of epithelium in periapical granulomas and the mechanism of cavitation in apical dental cysts in man. *Arch Oral Biol* 1974;19:1177–9.
18. Kubota Y, Ninomiya T, Oka S, Takenoshita Y, Shirasuna K. Interleukin-1 alpha-dependent regulation of matrix metalloproteinase-9 (MMP-9) secretion and activation in the epithelial cells of odontogenic jaw cysts. *J Dent Res* 2000;79:1423–30.
19. Motamedi MHK, Talesh KT. Management of extensive dentigerous cysts. *Br Dent J* 2005;198:203–6.
20. Tucker WM, Pleasants JE, MacComb WS. Decompression and secondary enucleation of a mandibular cyst: report of case. *J Oral Surg* 1972;30:669–73.
21. Marker P, Brondum N. Treatment of large odontogenic keratocysts by decompression and later cystectomy. *Oral Surg Oral Med Oral Pathol* 1996;82:122–31.
22. Nakamura N, Mitsuyasu T, Mitsuyasu Y, et al. Marsupialization for odontogenic keratocysts: long-term follow-up analysis of the effects and changes in growth characteristics. *Oral Surg Oral Med Oral Pathol Endod* 2002;94:543–53.
23. Pogrel MA, Jordan RCK. Marsupialization as a definitive treatment for the odontogenic keratocyst. *J Oral Maxillofac Surg* 2004;62:651–5.
24. Blanas N, Freund B, Schwartz M, Furst IM. Systematic review of the treatment and prognosis of the odontogenic keratocyst. *Oral Surg Oral Med Oral Pathol Endod* 2000;90:553–8.